Group A Streptococcal Puerperal Sepsis Preceded by Positive Surveillance Cultures

Karen R. Stefonek, BSN, MPH, Linda L. Maerz, MD, Michael P. Nielsen, MD, Richard E. Besser, MD, and Paul R. Cieslak, MD

Oregon Health Division, Portland, Oregon; Willamette Falls Hospital, Oregon City, Oregon; and the Childhood and Respiratory Diseases Branch, Division of Bacterial and Mycotic Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia.

BACKGROUND: Screening of pregnant women for vaginal and rectal carriage of group B streptococci may also identify group A streptococcal carriers. The clinical significance of prenatal group A streptococcal carriage is unknown.

CASES: Two women developed group A streptococcal puerperal sepsis after delivery at one hospital 15 months apart. The first patient required hysterectomy and suffered complications including subcapsular hepatic hematoma, pleural effusion, and prolonged ileus. She recovered after a 35-day hospitalization. The second patient had endometritis and recovered. Both had had group A streptococci isolated from vaginal and rectal cultures taken for prenatal group B streptococcal screening. The acute sepsis isolates were both M-type 28, but pulsed-field gel electrophoresis determined that the strains were unrelated.

CONCLUSIONS: Finding group A streptococci on prenatal culture may presage serious postpartum infection. (Obstet Gynecol 2001;98:846–8. © 2001 by the American College of Obstetricians and Gynecologists.)

The group A streptococcus holds a secure place in public health history as the cause of epidemic puerperal sepsis. Once the scourge of childbirth, group A streptococcus has in recent decades been an uncommon agent of postpartum infection.[1,2] More recently, group B streptococci have become the leading cause of peripartum infections,[3] and measures to reduce their incidence, such as screening pregnant women for colonization with group B streptococci and intrapartum prophylaxis for carriers, are being implemented.

In this report we describe two cases of postpartum group A streptococcal puerperal sepsis in women who had had group A streptococcal vaginal or rectal colonization documented before delivery when screening cultures for group B streptococci were obtained.

CASES

Case 1

A previously healthy multiparous woman presented to her obstetrician's office with shortness of breath, diaphoresis, and malaise. Four days earlier, induction of labor and episiotomy had been followed by uncomplicated vaginal delivery of her third child. Uterine bleeding after delivery had been treated with methergine. She had been discharged on the first postpartum day with her healthy newborn.

On readmission, her blood pressure was 94/70 mmHg, her temperature was 37.4C, and she had a sinus tachycardia with a rate of 136 beats per minute. Her white blood cell count was 16.2×10^9/L with a left shift, hematocrit was .44%, platelet count 170×10^9/L, bilirubin 29.1 μmol/L (1.7 mg/dL), serum glutamicoxaloacetic-transaminase (aspartate aminotransferase) 43 IU/L, blood urea nitrogen 27.7 mmol/L (55 mg/dL), creatinine 132.6 μmol/L (1.5 mg/dL), prothrombin time (PT) 12.6 seconds, partial thromboplastin time (PTT) 30.9 seconds, International Normalized Ratio 1.1, fibrinogen 22.1 μmol/L, and D-dimers were present. She had no skin rash.

Blood was obtained for culture, and antibiotic therapy was initiated with intravenous cefotetan. On hospital day 2, abdominal and pelvic computed tomography scans identified diffuse ascites, which, when tapped, revealed 3+ white cells and gram-positive cocci. Surgical abdominal exploration revealed a boggy, enlarged uterus covered with whitish exudate and bilateral enlarged, necrotic-appearing ovaries with multiple abscesses. Two liters of thin, bright yellow pus were drained from the peritoneal cavity, and a total abdominal hysterectomy and bilateral salpingo-oophorectomy were performed. The postoperative diagnosis was pelvic and abdominal sepsis secondary to myometritis with intraperitoneal abscess formation. On pathologic examination, the excised uterus showed a postpartum endometrium consisting almost entirely of purulent, hemorrhagic, necrotic debris pervading the myometrium with fibrin thrombi present in many endomyometrial vessels. The cervix and endocervix were heavily inflamed, and the ovaries had large confluent abscesses;

Address reprint requests to: Karen R. Stefonek, BSN, MPH, Oregon Health Division, 800 NE Oregon Street, Suite 772, Portland, OR 97232; E-mail: karen.r.stefonek@state.or.us.

This surveillance was made possible by the Emerging Infections Project/Active Bacterial Core Surveillance, a cooperative agreement between the Department of Human Services, Oregon Health Division, and the Centers for Disease Control and Prevention.

We gratefully acknowledge the contributions of Diane Davis, RN, BS, CIC, and Carole Eberle, MT, in investigating these cases; Piotr Kramarz, MD, PhD, for translating Polish-language literature; Bernard Beall, PhD, and Alma Franklin, for the isolate typing; and John A. Elliott, PhD, for subjecting the isolates to pulsed-field gel electrophoresis.

0029-7844/01/$20.00
PII S0029-7844(01)01611-8

abscesses were also present, but to a lesser degree, in the fallopian tubes. No retained products of conception were identified.

Ampicillin/sulbactam and clindamycin were given during the operation. Postoperatively, she was maintained on the ventilator for respiratory support and treated with vancomycin, metronidazole, and cefotetan. Vaginal cultures taken during the initial surgical procedure grew group A streptococci, *Escherichia coli* and *Bacteroides fragilis*. Cultures of blood and peritoneal fluid yielded group A streptococci susceptible to penicillin (minimum inhibitory concentration [MIC] 0.03 μg/mL) and resistant to clindamycin (MIC >2 μg/mL), erythromycin (MIC >4 μg/mL), and tetracycline (MIC 128 μg/mL). Uterine cultures were not done. With final confirmation of group A streptococci on hospital day 5, vancomycin was discontinued and penicillin G added. Her hospitalization was complicated by development of a subcapsular hepatic hematoma, which required surgical exploration on day 2 after the initial surgery, a right-sided pleural effusion, and a prolonged ileus. The patient was extubated on day 14, left the intensive care unit on day 21, and was discharged on hospital day 35, having had a total of 33 days of antibiotic therapy. Her newborn remained free of related infection. Review of prenatal records disclosed that group A streptococci had been isolated from a vaginal and rectal culture obtained 3 weeks before delivery as part of recommended practices for the prevention of group B streptococcal neonatal disease. No antibiotics had been prescribed based on this report of prenatal group A streptococci colonization.

Case 2

A woman presented to the hospital for assessment of early active labor at 37½ weeks' gestation. She had twice delivered healthy infants at 37 weeks' gestation, and had had two miscarriages: one at 6 and one at 10 weeks' gestation. After artificial rupture of membranes and administration of epidural anesthesia, her third child was delivered vaginally. Delivery was complicated only by a first-degree labial laceration that did not require repair.

During the first postpartum day, the patient suffered from exhaustion, uterine cramping, and nearly constant abdominal pain, which was unrelieved with naproxen sodium or acetaminophen with codeine. Her uterus was tender and palpable 3 cm below the umbilicus. After an episode of the chills, her oral temperature was 37.5C, her pulse was 72 beats per minute, her respirations were 16 per minute, and her blood pressure was 108/68 mmHg. Her white blood cell count was 4.9×10^9/L with a left shift, hematocrit was .32%, and platelet count 138×10^9/L. Urinalysis was normal. On postpartum day 2, her white blood cells rose to 8.1×10^9/L with a left shift, and she was afebrile. Before starting intravenous cefotetan, blood was drawn for culture. It subsequently yielded group A streptococci susceptible to penicillin (MIC <0.03 μg/mL), clindamycin (MIC < 0.25 μg/mL), levofloxacin (MIC <2 μg/mL), and tetracycline (MIC <2 μg/mL). On postpartum day 3, she was afebrile, had less abdominal tenderness, and her perineum was healing. She was discharged on postpartum day 4. Her newborn was discharged with her without evidence of infection. She completed 10 days of oral clavulanic acid plus amoxicillin. One month after discharge, she presented for outpatient follow-up with vaginal drainage, which, when cultured, grew group A streptococci with susceptibilities similar to the earlier blood cultures. She was treated with an additional 10 days of cephalexin, and the drainage resolved. Included in her postpartum medical record was the culture report from a vaginal swab obtained 11 days before delivery as part of routine practice for the prevention of group B streptococcal neonatal disease: it had yielded group A streptococci. No antibiotic treatment had been given after this screening result.

These two cases were delivered in the same hospital 15 months apart. The acute infection isolates were typed by sequencing the variable portion of the M-protein gene (*emm* typing). Pulsed-field gel electrophoresis, performed using previously described methods,[4] was used to determine the relatedness of the isolates. Sequence from both isolates demonstrated M-type 28; however, pulsed-field gel electrophoresis indicated that the strains were unrelated. Isolates from the prenatal screening cultures were not available from either case for comparison with the isolates obtained during the episodes of sepsis.

DISCUSSION

These are, according to our search of the literature, the first reported cases of group A streptococcal puerperal sepsis after documentation of vaginal and rectal colonization by this organism on prenatal culture. Using PubMed MEDLINE, we conducted an initial literature search of the years 1960–2001 (all journals and all languages). A more focused search for prior case reports of this type was from 1992 to 2001, again using PubMed MEDLINE (all journals and all languages). Search terms included "puerperal infection," "puerperal sepsis," "endometritis," "perinatal infection," "perinatal sepsis," "colonization," "group A streptococcus," and "streptococcus pyogenes." Historically, group A streptococcus was a common cause of puerperal infection, the organism having been carried from patient to patient on the hands of health care providers or transmitted directly from a colonized provider; indeed, outbreaks are still

occasionally reported.[5,6] We agree with recommendations for enhanced surveillance with identification of presumed nosocomial group A streptococcal sepsis,[6] but an endogenous infection source may warrant equal consideration. Although it is possible that our patients acquired their group A streptococci nosocomially, it seems more likely that their infections were born of pre-existing colonization. The incidence of such sporadic group A streptococci infection during the puerperium is unknown. Groups C and G streptococci have also caused significant puerperal infections, although their incidence is likewise difficult to discern from published studies.[7,8]

The American College of Obstetricians and Gynecologists, American Academy of Pediatrics, and the Centers for Disease Control have endorsed prevention of perinatal group B streptococci infections using either screening-based or risk-based strategies. The screening-based approach entails the culturing of all pregnant women for group B streptococci colonization at 35–37 weeks' gestation, so that transmission of this organism to newborns can be prevented by intrapartum penicillin. Because typical screening-based procedures identify them, it is inevitable that non-group B β-hemolytic streptococci will also be identified during these searches for group B streptococci. In a recent study, 0.03% of pregnant women screened at 35–37 weeks were found to have vaginal colonization with *S pyogenes* before delivery.[9]

At present, data are insufficient to make a recommendation regarding management of non-group B streptococcal colonization identified before delivery. However, these cases demonstrate that the finding of group A streptococci on prenatal culture may portend serious postpartum infection, and they lend weight to the suggestion of Davies et al, that microbiology laboratories should inform clinicians of any β-hemolytic streptococcus identified by prenatal screening cultures.[10] More precise information is needed about the risk associated with prenatal vaginal or rectal group A streptococci colonization and about the potential effectiveness of peripartum antibiotic prophylaxis against group A streptococcal puerperal sepsis.

REFERENCES

1. Silver RM, Heddleston LN, McGregor A, Gibbs RS. Life-threatening puerperal infection due to group A streptococci. Obstet Gynecol 1992;79:894–6.
2. Nathan L, Peters MT, Admed AM, Leveno KJ. The return of life-threatening puerperal sepsis caused by group A streptococci. Obstet Gynecol 1993;169:571–2.
3. Edwards MS, Baker CJ. Group B streptococcal infection. In: Remington J, Klein JO, eds. Infectious diseases of the fetus and newborn infant. 5th ed. Philadelphia: WB Saunders, 2000:1091–156.
4. Cockerill FR III, MacDonald KL, Thompson RL, Roberson F, Kohner PC, Besser-Wiek J, et al. An outbreak of invasive group A streptococcal disease associated with high carriage rates of the invasive clove among school-aged children. JAMA 1997;277:38–43.
5. Viglionese A, Nottebart VF, Bodman HA, Platt R. Recurrent group A streptococcal carriage in a health care worker associated with widely separated nosocomial outbreaks. Am J Med 1991;91(Suppl):329–33.
6. Centers for Disease Control and Prevention. Nosocomial group A streptococcal infections associated with asymptomatic health-care workers–Maryland and California, 1997. MMWR Morb Mortal Wkly Rep 1999;48:163–6.
7. Vartian C, Lerner PI, Shlaes DM, Gopalakrishna KV. Infections due to Lancefield group G streptococci. Medicine 1985;64:75–88.
8. Salata RA, Lerner PI, Shlaes DM, Gopalakrishna KV, Wolinsky E. Infections due to Lancefield group C streptococci. Medicine 1989;68:225–39.
9. Mead PB, Winn MC. Vaginal-rectal colonization with group A streptococci in late pregnancy. Infect Dis Obstet Gynecol 2000;8:217–9.
10. Davies HD, McGeer A, Schwartz B, Green K, Cann D, Simor AE, et al. Invasive group A streptococcal infections in Ontario, Canada. N Engl J Med 1996;335:547–54.

Received May 26, 2001. Received in revised form July 26, 2001. Accepted August 2, 2001.